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PSYCHOLOGICAL KNOWLEDGE IN COURT

PTSD, Pain, and TBI

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Comorbid Chronic Pain and Posttraumatic Stress Disorder Across the Lifespan: A Review of Theoretical Models

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Chronic pain is often associated with functional, psychological, and social problems that can have a significant negative impact on a person's quality of life. Substantial literature currently exists documenting the relationship between chronic pain and commonly co-occurring disorders such as substance abuse (Brown et al., 1996), depression (Banks & Kerns, 1996), and anxiety disorders (Asmundson et al., 1996). Importantly, interest in the relationship between chronic pain and these disorders has expanded the field of pain research, has improved our understanding of how these conditions may interact with one another, and has contributed to improvements in pain management.

Pain is often related to naturally occurring degenerative changes in the body that develop gradually over time; however, some pain conditions may develop secondary to injury related to traumatic life events such as motor vehicle accidents, occupational injuries, or military combat. This has led to a growing interest in the relationship between pain and Posttraumatic Stress Disorder (PTSD), as clinical practice and research indicate that the two disorders frequently co-occur and may interact in such a way as to negatively impact the course and outcome of treatment of either disorder. Several theoretical models and potential mechanisms have been proposed to explain the relationship between chronic pain and PTSD. Despite this recent interest in studying pain and PTSD, no empirical studies have been conducted to test theoretical models explaining the comorbidity of these two disorders, and no well-controlled studies have investigated the efficacy of tailoring treatments for individuals with these disorders. Such studies have the potential to advance theory development and improve treatment efficacy.

The primary aim of this chapter is to provide a critical review and synthesis of the existing literature investigating the relationship between chronic pain and PTSD. The chapter begins with a presentation of the diagnostic criteria,

prevalence, and theoretical models of chronic pain and PTSD. Research is then presented describing the co-occurrence of the two disorders, and several theoretical models are highlighted that may serve to explain the similar mechanisms by which these two disorders may be maintained. This chapter then addresses how comorbid chronic pain and PTSD may present differently in youth, with consideration of how theoretical models of the comorbidity of pain and PTSD may be modified to incorporate developmental factors. Furthermore, the chapter helps to explicate how the experience of comorbid chronic pain and PTSD can vary from childhood to adulthood. Finally, the chapter closes with a section on implications for treatment as well as a call for continued research to further refine the models reviewed.

1. Chronic Pain

Although pain is typically a transient experience, for some people pain persists past the point where it is considered adaptive and results in emotional distress, impaired occupational and social functioning, and increased use of health care system resources (Benedetto et al., 1998). For example, individuals with chronic pain often report that pain interferes with their ability to engage in occupational, social, or recreational activities. Their inability to engage in these reinforcing activities may contribute to increased isolation, negative mood (e.g., feelings of worthlessness and depression), and physical deconditioning, which, in turn, can contribute to their experience of pain. Over time, these types of deleterious cognitive and behavioral patterns can become more resistant to change.

Pain is currently defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or is described in terms of such damage. Pain that persists for an extended period of time (i.e., months or years), that accompanies a disease process, or that is associated with a bodily injury that has not resolved over time may be referred to as "chronic" pain (IASP Task Force on Taxonomy, 1994). Pain is one of the most common complaints made to primary care providers (Gureje et al., 1998) and has significant implications for health care costs. For example, a recent study estimated that the total health care expenditures for back pain alone reached over \$90.7 billion in 1998 (Xuemei et al., 2004).

Biopsychosocial models suggest that pain is not just a biological process involving the transmission of sensory information about tissue damage to the brain, but is the product of the interactions among biological, psychological, and social factors. All of these factors have an impact on a person's experience of pain, including pain intensity, duration, and its consequences. Models have been proposed to incorporate the many factors that might contribute to the development of chronic pain. For example, Kerns et al. (2002a) proposed a cognitive-behavioral, transactional model of chronic pain, emphasizing the importance of social support and the family in the

development and maintenance of chronic pain. Research supports the hypothesis that positive attention from a spouse in response to a patient's expression of pain is associated with higher levels of pain (Kerns et al., 1990), higher frequency of observed pain behaviors (e.g., grimacing, bracing, and distorted ambulation) (Romano et al., 1992), and reports of greater disability and interference (Turk et al., 1992). In addition, there is evidence that a high frequency of negative responding to pain is reliably associated with severity of affective distress (Kerns et al., 1990).

Vlaeyen and Linton (2000) proposed a cognitive-behavioral, fear-avoidance model of chronic pain to explain the role of fear and avoidance behaviors in the development and maintenance of chronic pain and related functional limitations. According to this model, there are two opposing responses an individual may have when experiencing pain. One response is that an individual may consider pain to be nonthreatening and consequently engage in adaptive behaviors that promote the restoration of function. In contrast, pain may be interpreted as overly threatening, a process called "catastrophizing." Vlaeyen and Linton (2000) proposed that catastrophizing contributes to a fear of pain and may lead to avoidance of activities that could elicit pain, guarding behaviors, and hypervigilance to bodily sensations. Consistent with principles of operant reinforcement, as activities are avoided and feelings of fear subside, avoidance behaviors are positively reinforced. As an individual becomes more depressed and inactive, the cycle of pain is fueled even further, and fear and avoidance is further increased. Thus, avoidance has the potential to increase disability and negative mood and ultimately contribute to the experience of pain. Research supports a relationship between fear-avoidance and chronic pain (Asmundson & Taylor, 1996; Crombez et al., 1999).

2. Posttraumatic Stress Disorder

Posttraumatic Stress Disorder can occur following exposure to an event that is, or is perceived to be, threatening to the well-being of oneself or another person. The distinctive profile of symptoms in PTSD include (1) exposure to a traumatic event that involved the threat of death or serious injury (Criterion A), (2) re-experiencing the event in the form of intrusive thoughts, nightmares, flashbacks to the traumatic event, and psychophysiological reactivity to cues of the traumatic event (Criterion B), (3) avoidance of thoughts, people, and places that resemble the traumatic event, emotional numbing, and an absence of emotional attachments (Criterion C), and (4) symptoms of hyperarousal, including heightened startle sensitivity, sleep problems, attentional difficulties, hypervigilance, and the presence of irritability and anger (Criterion D) (American Psychiatric Association, *Diagnostic and statistical manual of mental disorders* 1994). High levels of anxiety, depression, panic, and substance abuse are frequently observed in individuals with PTSD (Keane & Wolf, 1990).

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It has been estimated that the prevalence of PTSD in the United States is 6% in males and 12% in females (Kessler et al., 1995). However, exposure to traumatic events has been estimated to be as high as 70% of the adult population (Norris, 1992). These numbers suggest that PTSD is among the most frequent of psychological disorders, ranking behind substance abuse and depression. In the most comprehensive study of the effects of war on its combatants, the National Vietnam Veterans Readjustment Study (Kulka et al., 1990) found a lifetime rate of PTSD of 30% and a current rate of 15%.

Although many people may be exposed to the same potentially traumatic event, not everyone will develop PTSD. The literature suggests that although the experience of a traumatic event certainly contributes to the development of PTSD, other factors also play an important role (Keane & Barlow, 2002; McNally, 2003). Psychosocial factors such as personal hardiness, structural and functional social support, and stressful life events have all been identified as having direct effects on the development of PTSD, and it is believed that these effects may differ for men and women (King et al., 1998). Research also suggests that peritraumatic psychological processes such as dissociation are strong predictors of PTSD (Ozer et al., 2003). Although there is some preliminary evidence to suggest that there may be a genetic or hereditary component to the development of PTSD (Davidson et al., 1991), most studies conducted in this area have had methodological limitations, and continued research with improved experimental design is needed before firm conclusions can be drawn. Thus, it appears that psychosocial factors and personal vulnerabilities may play a significant role in influencing the impact a traumatic event has on a person's life.

A number of theories have been proposed for the development of PTSD (Brewin & Holmes, 2003). Behavioral conceptualizations of PTSD are based on the two-factor learning theory (Mowrer, 1960). The theory proposes that fear is first learned via classical conditioning in which neutral stimuli present in the traumatic situation acquire fear-eliciting properties through their association with elements of the traumatic situation that arouse fear. The second stage is marked by avoidance behavior that minimizes the contact time with the conditioned cues, thus impairing the extinction of the learned fear. Keane et al. (1985) extended this model to humans and proposed that an extreme stressor, such as a traumatic event, acts as an unconditioned stimulus that can create learned associations with internal and external cues (e.g., sights, sounds, or smells) that are present at the time of the stressor. After these conditioned associations are established, previously neutral cues can then elicit strong autonomic and physiological responses that are similar to those experienced at the time of the stressor. These responses may be so aversive that the individual begins to avoid the triggering cues as a way to decrease his or her own fearful reactions (Keane et al., 2000).

Cognitive and information-processing theories of PTSD have been developed from Lang's (1979) bio-informational theory of emotion. According to Lang

(1979), "fear networks" store representations in memory of anxiety-provoking events and contain information about a feared stimulus or situation, the person's cognitive, psychophysiological, and behavioral response to the stimuli or situation, and information about the meaning of the feared stimuli. Anxiety disorders develop when the fear network contains faulty connections and information that does not truly represent the state of the world. Foa and Kozak (1986) have proposed that when compared to other anxiety disorders, the size of the fear network in PTSD is larger, the network is more easily activated, and the affective and physiological response elements of the network are more intense.

In addition to these early theoretical approaches, a number of more recent theories regarding the etiology of PTSD have been proposed. Several of these recent theories are described in detail by Brewin and Holmes (2003). Among those reviewed are Foa and Rothbaum's (1998) emotional processing theory, Brewin et al. (1996) dual representation theory, and Ehlers and Clark's (2000) cognitive theory. It should be noted that there is a high degree of overlap between these recent models of PTSD, with all models incorporating a wide range of findings on the importance of factors affecting encoding, alterations in memory functioning, appraisals, coping strategies and cognitive styles, and the importance of prior beliefs and trauma exposure. They differ significantly, however, in their accounts of how trauma impacts on memory, the processes whereby changes are brought about in memory, and how these changes are related to recovery (Brewin & Holmes, 2003).

3. Comorbid Pain and PTSD

Several studies have assessed the co-occurrence of PTSD and chronic pain symptoms. For example, using DSM-III-based diagnostic criteria, one study found that 10% of patients referred to a chronic pain clinic met criteria for PTSD (Benedikt & Kolb, 1986), whereas another study reported that 9.5% met criteria for "posttraumatic pain syndrome" (Muse, 1986). A review of the more recent literature suggests that between 20% and 34% of patients referred for the treatment of chronic pain have significant PTSD symptomatology or are diagnosed with PTSD (Asmundson et al., 1998; Geisser et al., 1996). Asmundson et al. (1998) performed a study to assess the extent to which work-related injuries were associated with PTSD. Assessments were conducted on 139 injured workers with chronic pain who were referred to a rehabilitation program. The results indicated that 34.7% of the sample reported PTSD symptoms. Research indicates that rates of PTSD in patients for which pain is secondary to motor vehicle accidents (MVAs) range from 30% to 50% (Chibnall & Duckro, 1994; Hickling & Blanchard, 1992; Hickling et al., 1992; Taylor & Koch, 1995). Studies suggest that from 24% to 47% of fibromyalgia patients attribute the onset of their symptoms to a physical injury associated with an MVA (Sherman et al., 2000; Turk et al., 1996).

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Geisser et al. (1996) examined the self-report of pain, affective distress, and disability in pain patients with and without PTSD symptoms. Their results indicated that patients with accident-related pain and high PTSD symptoms reported higher levels of pain and affective distress relative to patients with accident-related pain and without PTSD, or nonaccident-related pain.

Studies examining the prevalence of chronic pain in individuals with a primary diagnosis of PTSD have reported even higher coprevalence rates. Amir et al. (1997) examined a sample of 29 PTSD patients and found the prevalence of fibromyalgia syndrome to be 21%. McFarlane et al. (1994) reported that in a sample of PTSD patients reporting physical symptoms, pain was the most common physical complaint (45% reported back pain and 34% reported headaches). White and Faustman (1989) performed a review of discharge summaries of 543 veterans treated for PTSD to assess the frequency and nature of medical problems. Their results indicated that 60% had an identified medical problem, and one in four showed some type of musculoskeletal or pain problem. Beckham et al. (1997) investigated chronic pain patterns in Vietnam veterans with PTSD. A sample of 129 combat veterans with PTSD completed self-report questionnaires assessing PTSD symptoms and current pain status. Eighty percent of the sample reported the presence of a chronic pain condition. In addition, increased levels of PTSD re-experiencing symptoms were associated with increased pain and disability.

There could be several reasons for the high degree of variability in the prevalence rates reported in these studies of comorbid pain and PTSD. An inspection of these studies indicates that there were differences in the populations sampled (e.g., pain clinics vs. PTSD clinics). In addition, some of the studies cited based their coprevalence rates on diagnostic criteria from the DSM-III, whereas other studies based their prevalence rates on more recent diagnostic criteria. Further, assessment instruments utilized in the above studies varied widely; for example, some studies relied on the retrospective review of medical records to diagnose PTSD or chronic pain, whereas other studies utilized more sophisticated diagnostic and assessment instruments such as the CAPS (for PTSD) (Blake et al., 1990) or the WHYMPI (for pain) (Kerns et al., 1985). Thus, given the limitations of previous research, it is difficult to determine exact figures regarding the prevalence of comorbid pain and PTSD. Future studies should include state-of-the-art diagnostic measures to gain an accurate picture of the coprevalence of these conditions.

The co-occurrence of chronic pain and PTSD may have implications in terms of an individual's experience of each condition. Research indicates that patients with chronic pain related to trauma or PTSD experience more intense pain and affective distress (Geisser et al., 1996; Toomey et al., 1994), higher levels of life interference (Turk & Okifuji, 1996), and greater disability (Sherman et al., 2000) than pain patients without trauma or PTSD. For example, Chibnall and Duckro (1994) examined a sample of chronic post-traumatic headache patients and found that patients with PTSD and pain had higher levels of depression and suppressed anger than pain patients

without PTSD. Tushima and Stoddard (1990) found that patients with post-traumatic headache reported more frequent pain and had a poorer prognosis than did nontraumatic headache patients. Sherman et al. (2000) found that in a sample of 93 treatment-seeking fibromyalgia patients, those who experienced PTSD-related symptoms reported significantly greater levels of pain, life interference, emotional distress, and inactivity than did patients who did not report PTSD-like symptoms. Over 85% of the sample with significant PTSD-like symptoms demonstrated significant disability compared to 50% of the patients without significant PTSD-like symptoms. Sherman and colleagues suggested that clinicians should address these PTSD symptoms in pain treatment, as failure to attend to them could limit successful outcomes. Thus, taken together, these studies suggest that the presence of both PTSD and chronic pain can increase patients' overall symptom severity.

4. Possible Explanations of the Relationship Between Chronic Pain and PTSD: Examination of Theoretical Models

The high rate of comorbidity and symptom overlap between chronic pain and PTSD suggests that the two disorders might be related in some way. Clearly, this review of studies establishing the co-occurrence between pain and PTSD does not provide an explanation of the mechanisms by which they are linked. Although theoretical models have been proposed to account for the co-occurrence of pain and PTSD, these theoretical models have yet to be tested. However, there are numerous factors presented in these models that might be implicated in the etiology and maintenance of both conditions. In several recent articles (Asmundson et al., 2002; Otis et al., 2003; Sharp & Harvey, 2001), theories hypothesizing the relationship between chronic pain and PTSD were reviewed and several potential mechanisms of action were described. For clarity, each model will be reviewed in turn.

4.1. *Mutual Maintenance Model*

According to Sharp and Harvey's (2001) Mutual Maintenance Model of chronic pain and PTSD, seven specific factors are identified by which mutual maintenance of chronic pain and PTSD could occur. Although many of the factors described by Sharp and Harvey have not been empirically investigated, they are useful in that they may serve to stimulate more critical examinations of the comorbidity of chronic pain and PTSD and provide several possible directions for future research. These factors include (1) attentional biases (which may cause patients to attend to threatening or painful stimuli), (2) anxiety sensitivity (which may contribute to a vulnerability to catastrophize about pain or trauma), (3) pain as a reminder of trauma (triggering an arousal response, avoidance of the cause of pain, and any memories of the

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trauma), (4) avoidance (may be adopted as a means to minimize pain and disturbing thoughts of the trauma), (5) fatigue and lethargy (associated with depression), (6) general anxiety, and (7) cognitive demands (caused by symptoms of pain and PTSD, may limit the use of adaptive coping strategies). Several other models have been developed by other researchers that have also included one or more of these factors to help explain the relationship between chronic pain and PTSD. Descriptions of these models, including existing empirical research on the factors maintaining pain and PTSD, will be presented below.

4.2. *Shared Vulnerability Model*

Anxiety sensitivity is the fear of arousal-related sensations, arising from beliefs that these sensations have harmful consequences. Evidence suggests that anxiety sensitivity contributes to or amplifies the intensity of emotional reactions, particularly those with an anxiety component such as panic (Taylor, 2003). For example, persons with high anxiety sensitivity may become anxious when confronted with a feared situation as a result of their experience and as a result of their fearful interpretation of the physiological sensations of anxiety (e.g., racing heart, perspiration, shortness of breath). Asmundson et al. (2002) proposed a shared vulnerability model of chronic pain and PTSD in which anxiety sensitivity is a predisposing factor contributing to the development of both conditions. According to this model, a person with high levels of anxiety sensitivity is likely to become fearful in response to physical sensations such as heart pounding and breathlessness, thinking that these symptoms may signal impending doom. When people with high anxiety sensitivity encounter either a traumatic stressor or pain (or both), they are believed to respond with more fear than those with low anxiety sensitivity. Thus, the tendency to respond with fear to physical symptoms of anxiety is seen as a shared vulnerability contributing to the development of either disorder. In the case of PTSD, the degree of alarm caused by the stressor is combined with the alarm of physiological sensations to further exacerbate the emotional reaction, thereby increasing the risk of developing PTSD. In the case of chronic pain, anxiety sensitivity heightens fear and avoidance of activities that could induce pain, which further increases the chances that pain will be maintained over time.

Clinical pain research supports a relationship between anxiety sensitivity and pain. For example, Asmundson and Norton (1995) found that patients with higher anxiety sensitivity were more likely to experience greater anxiety and fear of pain, more negative affect, and greater avoidance of activities. Asmundson and Taylor (1996) found that anxiety sensitivity directly increased fear of pain; however, anxiety sensitivity indirectly influenced avoidance and escape behaviors through fear of pain. More recently, Zvolensky et al. (2000) evaluated anxiety sensitivity, depression, and pain severity as potential predictors of pain-related fear in a heterogeneous

chronic pain population. Their findings indicated that anxiety sensitivity, as measured by the Anxiety Sensitivity Index (ASI; Reiss et al., 1986) was a better predictor of fear of pain and anxiety about pain than other relevant variables. Finally, Greenberg and Burns (2003) examined pain-related anxiety in a group of chronic musculoskeletal pain patients who underwent an experimentally induced pain induction procedure (i.e., cold pressor) and had to complete mental arithmetic tasks. Their results indicated that almost all of the effects of pain anxiety on task responses were accounted for by anxiety sensitivity. Taken together, results of all of these studies support the hypothesis that anxiety sensitivity may represent a vulnerability factor in the development and maintenance of pain-related anxiety and avoidance behaviors.

Although the majority of studies on anxiety sensitivity indicate that it may play a role in the development of anxiety disorders such as panic (Barlow, 2002), there are also several studies that support the model of anxiety sensitivity as a vulnerability factor in PTSD. In a study that examined the presence of anxiety sensitivity in 313 individuals with anxiety disorders, Taylor et al. (1992) found that patients with PTSD were the second highest on the ASI measure, with patients diagnosed with panic being the highest. Lang et al. (2002) found that anxiety sensitivity was a significant predictor of PTSD symptoms in women. In another study, anxiety sensitivity was related to severity of PTSD symptoms, and a reduction in anxiety sensitivity after participation in a cognitive-behavioral therapy (CBT) treatment program was related to a reduction in PTSD symptoms (Fedorff et al., 2000). Thus, although preliminary research is supportive, additional research on the interaction of anxiety sensitivity and PTSD would help to clarify the role of anxiety sensitivity as a potential vulnerability factor contributing to the development of PTSD.

4.3. *Fear-Avoidance Model*

Building upon Vlaeyen and Linton's (2000) cognitive-behavioral fear-avoidance model of chronic pain, Norton and Asmundson (2003) recently proposed a fear-avoidance model of chronic pain that places a larger emphasis on the contributions of physiological activity and arousal (e.g., increased blood flow, heart rate, or muscle tension) in the perpetuation of fear and avoidance related to pain. There are two primary amendments to the model: (1) Physiological sensations could increase avoidance by aggravating damaged or weakened tissues and increasing pain and (2) physiological sensations of arousal could be misinterpreted as being pain related. These misinterpretations may be influenced by an individual's tendency to respond with fear to sensations that are anxiety provoking (e.g., individuals with high anxiety sensitivity). For example, an individual with a tendency to be hypervigilant to pain-related bodily sensations may interpret the physiological sensations as evidence of impending harm or pain, which would reinforce fears and beliefs that activities will be painful, and thus reinforce avoidance.

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Avoidance also plays a significant role in models of chronic pain and PTSD. In chronic pain, avoidance can contribute to decreased feelings of self-efficacy related to pain, negative expectations and beliefs about an individual's ability to cope with pain, and increased disability (Waddell et al., 1993). The avoidance of reinforcing activities and social situations can contribute to affective distress, which can further exacerbate the experience of pain (Romano & Turner, 1985). Similarly, for a person with PTSD, fear of re-experiencing disturbing thoughts of events and avoidance of reminders associated with the trauma are core components of this disorder. This fear and avoidance can serve to prevent effective processing of the traumatic event and may lead to the maintenance of intrusive symptoms of re-experiencing the trauma and increased arousal (Keane et al., 2000).

Catastrophizing is another factor that has been implicated in Vlaeyen and Linton's (2000) fear-avoidance model, and catastrophizing may also play a significant role in the development and maintenance of both chronic pain and PTSD. For example, pain research indicates that patients who use passive and maladaptive coping strategies such as catastrophizing are more likely to report greater pain and disability than patients who use active and adaptive coping strategies such as exercise, ignoring pain, and positive self-coping statements (Boothby et al., 1999; Jensen et al., 1999; Severeijns et al., 2001). In a recent study by Jensen et al. (2001), it was found that decreases in catastrophizing and the belief that one is disabled and increases in perceived control over pain were associated with decreases in pain, disability, and depression in a sample of 141 patients receiving multidisciplinary treatment for chronic pain. Likewise, for individuals with PTSD, catastrophizing thoughts regarding what might occur in feared situations may cause increased avoidance, unrealistic expectations, and increased distress regarding resumption of everyday activities (Asmundson et al., 2002).

4.4. *Triple Vulnerability Model*

Otis et al. (2003) applied the Triple Vulnerability Model (Barlow, 2000, 2002) to explain the development of chronic pain and PTSD. According to the Triple Vulnerability Model of anxiety (Barlow, 2000, 2002), an integrated set of triple vulnerabilities need to be present for developing an anxiety disorder: a generalized biological vulnerability, a generalized psychological vulnerability based on early experiences of lack of control over salient events, and a more specific psychological vulnerability in which one learns to focus anxiety on specific situations. Whereas the Triple Vulnerability Model applies to the development of anxiety in general, Keane and Barlow (2002) proposed a model of the development of PTSD specifically. According to their model, a true or false alarm develops during exposure to situations that symbolize or resemble an aspect of a traumatic event. However, the experience of alarm or other intense emotions is not sufficient in and of itself for the development of PTSD. In order to develop PTSD, one must develop anxiety or the sense

that these events, including one's own emotional reactions to them, are preceding in an unpredictable and uncontrollable manner. Thus, when negative affect and a sense of uncontrollability develop, PTSD may emerge. Although this model implies that a psychological and biological vulnerability to develop the disorder exists, it has been found that anxiety is always moderated to some extent by variables such as the presence of adequate coping skills and social support (Keane & Barlow, 2002).

Although designed to describe the development of PTSD, this model could also relate to the development of chronic pain. For example, pain may have a biological basis and persons may have a genetic predisposition to develop certain pain conditions, as has been suggested in the case of headache (Larsson et al., 1995; Russell, 2004). Also, pain can be the result of a physical injury or the gradual deterioration of tissue over time. However, it has been consistently demonstrated that the presence and extent of physical pathology by itself is often not sufficient to account for the report of pain. For example, there is a low correlation between abnormal magnetic resonance imaging (MRI) and pain report, and many individuals with abnormal MRIs do not report impairment (Wood et al., 1995). Thus, just as a biological vulnerability is one risk factor in the development of anxiety, yet is not sufficient to cause an anxiety disorder; pain, too, may have a biological basis, but the presence of tissue damage or pathology alone is not sufficient to cause a chronic pain condition. Similar to the Triple Vulnerability Model developed for anxiety, a generalized psychological vulnerability may also be present prior to the development of a chronic pain condition. Numerous studies indicate that many chronic pain patients do experience perceptions of low social support, poorly developed coping skills, and failed past attempts to cope with stressful life events (e.g., job stressors, marital stressors), as well as perceptions of lack of control over life events (DeGood & Tait, 2001). More specifically, it is possible that for some people to develop a chronic pain condition, they must also develop a belief that the pain is preceding in an unpredictable and uncontrollable manner. When combined with a previous experience of coping poorly with a painful condition, this could contribute to decreased self-efficacy and low expectations of adaptively coping with future experiences of pain, which may constitute a specific psychological vulnerability to developing chronic pain. There are numerous studies that indicate that many chronic pain sufferers do, in fact, typically perceive a lack of personal control over their pain (Turk & Rudy, 1988). The relationship between perceived controllability and pain has been demonstrated in a variety of chronic pain syndromes, including migraine headache patients (Mizener et al., 1988), and low back pain patients and rheumatoid arthritis patients (Flor & Turk, 1988). When persons perceive their pain to be uncontrollable, feelings of low self-efficacy may develop, along with negative affect. Thus, a fear may develop of entering situations or performing activities in which pain could occur, leading to avoidance of situations in daily life. This avoidance will further fuel negative affect, feelings of uncontrollability, and

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low self-efficacy, resulting in increased disability. Similar to the PTSD literature, it has been found that pain is always moderated to some extent by variables such as the presence of adequate coping skills and social support (Kerns et al., 2002a, 2002b). Thus, whether the "alarm" is a trauma reminder or pain reminder, the development of a sense of uncontrollability could precede the development of both disorders.

5. Comorbidity of Chronic Pain and PTSD: Developmental Considerations

Although the models and empirical research presented earlier are almost entirely based on work with adults, it is important to consider that comorbid pain and PTSD could occur throughout one's life span. Although thorough reviews of the literature on pain and PTSD in children could certainly stand alone as separate chapters, some of the developmental issues to consider are presented briefly here in order to provide a broader scope regarding issues of comorbidity and how they may relate to youth. In the following section, developmental issues affecting PTSD and chronic pain in children are discussed, followed by a section highlighting potential ways that the theoretical models of the maintenance of the two disorders may be tailored to incorporate developmental factors.

6. PTSD in Children and Adolescents

Each year in the United States, more than 5 million children are exposed to some form of extreme traumatic stressor, such as natural disasters, physical abuse, sexual assault, MVAs, life-threatening illnesses, painful medical procedures, witnessing of community or domestic violence, sudden death of a parent or loved one, or traumatic injuries or illnesses (Pfefferbaum, 1997). More than 30% of those children exposed to an extreme traumatic stressor develop PTSD, and as a result they experience symptoms that may affect them physically, emotionally, cognitively, behaviorally, and socially (Perry, 1999). Similar to adults, children can develop PTSD in response to a range of traumatic stressors; however, children may express distress as a result of a trauma quite differently from adults, with the child's initial reaction to trauma including the possibility of disorganized or agitated behavior, such as crying, clinging, or hyperkinesis (Perrin et al., 2000). Furthermore, although children may re-experience a traumatic event just as adults do, young children may not be able to verbalize their thoughts or have the cognitive capacity to recall important aspects of the trauma. Nightmares involving the theme of the trauma are common in children of all ages, and young children often display signs of re-experiencing through vivid re-enactment of the trauma in the form of drawings, stories, and play (Scheeringa et al., 1995). Thus, although

children experience both Criterion A (the traumatic event) as well as Criterion B (re-experiencing), these symptoms may be observed differently in children than in adults. Children also experience Criterion C (persistent avoidance and numbing of general responsiveness), and avoidance of both external and internal trauma reminders are common in traumatized children and adolescents. Just as adults may avoid traumatic reminders, and this avoidance can be reinforced by significant others such as a spouse, children also may avoid traumatic reminders, and parents' reactions to the trauma can significantly influence children's reaction. For example, some parents may inadvertently (or even purposefully) reinforce children's avoidance of reminders of the trauma (e.g., allow the child to sleep with them rather than alone, allow the child to avoid school), whereas other parents will not permit such avoidance. Thus, as sequelae to trauma, children may lose interest in participating in significant activities that are part of normal development; this avoidance can further perpetuate the child's vulnerability to feelings of depression or anxiety. Furthermore, older children and adolescents may develop a view of life as being quite fragile after a traumatic event, leading to expectations of negative events happening to them in the future and leading to further avoidance of normal daily activities. Finally, similar to adults, children may also experience persistent symptoms of increased arousal (Criterion D). For example, somatic complaints such as headaches or stomachaches are commonly reported by children who have been traumatized, in addition to intense fears. Irritability and outbursts of anger are also common. As could be expected, children who have been traumatized often find it difficult to concentrate in school and may evidence inattentiveness or hyperactivity (Lyons & Adams, 1999). In addition, children may also report hypervigilance, overprotectiveness of caregivers, separation anxiety, and prolonged upset in response to loud noises and/or arguing (Steward & O'Connor, 1994). Given the differences between the presentation of PTSD in adults and children, assessment and treatment strategies must be developmentally tailored to address the many factors that may be specific to the age of the patient.

7. Chronic Pain in Children and Adolescents

Brief episodes of acute pain related to routine injuries and illnesses in childhood are common, with 15% of healthy school-aged children reporting brief episodes of pain (Chambliss et al., 2002). Children's typical responses to acute pain are usually short-lived, and normal activity is often quickly resumed, as is typically observed with adults. However, chronic pain in children, often associated with an underlying disease, a traumatic injury, or an ongoing trauma causing sustained injury, can result in unnecessary suffering of the child and family, disruption of the family routine, and restriction of the child's daily activities, thereby increasing the risk of long-term disability (Caffo & Belaise, 2003). In fact, chronic pain in childhood can often result in

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somatic and psychiatric dysfunction, with studies showing that children experiencing chronic pain are more likely than other children to complain of anxiety, to demonstrate hypochondriacal beliefs, to engage less frequently in social activities, and to experience higher levels of generalized anxiety (Campo et al., 2001). Chronic pain conditions in childhood may arise because of known injury (such as rheumatologic disease, sickle cell disease, or human immunodeficiency virus infection), or to traumatic injury (because of burns, physical abuse, or MVAs), whereas some chronic pain conditions in childhood may have less clear etiologies (e.g., chronic headache) (Chambliss et al., 2002). As a result of increased research over the past 20 years on chronic pain in children, we now understand that child pain, like adult pain, is not simply directly related to the extent of physical injury or level of tissue damage, but is influenced by many psychological factors that can modify the neural signals for pain and increase or decrease a child's distress. It has been suggested that children's pain is more "plastic" than that of adults, such that psychosocial factors could exert an even more powerful influence on children's pain perception than on adults' pain perception (McGrath & Hillier, 2002).

The presentation of chronic pain in children may also differ from that of adults, and there are numerous factors that may influence the child's experience of pain, including child factors (such as age, cognitive level, or temperament), cognitive factors (e.g., expectations about treatment efficacy), behavioral factors (e.g., child's distress responses, avoidance of activities), and emotional factors (anticipatory anxiety, depression) (McGrath & Hillier, 2002). Although some of these factors are stable for a child (e.g., temperament), other factors change progressively (e.g., age, cognitive level, physical state, and family learning). Child factors and situational factors (e.g., level of control over situation) may interrelate to shape how children generally interpret the various sensations caused by tissue damage. For example, as children grow, they learn ways to express pain and ways to cope with pain, and their experience is certainly shaped by their family, culture, and interactions with caregivers and peers. This notion is consistent with Melzack and Wall's (1965) definition of pain as a multidimensional and subjective experience, characterized by physiologic, affective, cognitive, behavioral, and cultural dimensions. Thus, even though the tissue damage for several children may be the same, certain factors specific to each child or to each child's environment can intensify pain and distress, trigger pain episodes, and prolong pain-related disability, whereas other factors may buffer the effects of the pain, enable the child to engage in healthy coping, and lessen distress. Thus, a thorough assessment is crucial to determine the extent to which cognitive, behavioral, emotional, or situational factors contribute to or buffer the pain experience for a child, with understanding that these factors are likely to vary between children and may even vary over time for the same child.

Children's ongoing physical growth may also play a role in their ability to recover more quickly than adults from injury. Pain behavior in children has

also been found to vary as a function of the child's developmental level. Older children will be able to describe the location, intensity, duration, and sensation of pain, whereas younger children may not be able to distinguish pain from other negative affective states (Tarnowski & Brown, 1999). Pain behavior in children has also been found to differ depending on the presence or absence of a caregiver during a painful medical procedure, with some studies finding that children whose mothers were present were more distressed, but that children prefer parents or caregivers to be present (Gonzalez et al., 1989). Parents' attitudes and expectations, their anxiety levels, and whether they are overly protective and reinforcing of dependence are variables that may affect children's ability to successfully cope. Also, parents may inadvertently cue and reinforce their child's distress, whereas others may promote coping by the child (Blount et al., 1991). Because of the number of parental variables that could influence child coping, there is need to assess characteristics of the parent, child, and parent-child interactions when assessing pain in children. Given the host of factors that may influence a child's experience of pain, it is not surprising that the treatment of pain in childhood requires an integrated approach, informed by the many factors that may influence a child's pain, including the family and cultural factors that might impact the child, and the child's current methods of coping with pain. Cognitive behavioral treatments for chronic pain in children should take these factors into consideration by giving children effective strategies that will lessen their pain and distress and help them return to developmentally appropriate activities.

8. Comorbid Chronic Pain and PTSD in Children and Adolescents

Given the range of symptoms experienced by youth experiencing PTSD and chronic pain, it is surprising that there has not been more extensive research specifically on children who experience comorbid chronic pain and PTSD. The majority of the research that has been conducted has focused on children who suffer traumatic injury (pain resulting from trauma or abuse). Stoddard and Saxe (2001) stated that traumatic injuries are the single largest cause of morbidity among children in the United States. In fact, 8.7 million children under the age of 15 are seen in emergency departments because of traumatic injuries resulting from automobile accidents, falls, violence, or sports-related injuries (Scheidt et al., 1995). More than 30% of children who experience a painful traumatic injury develop PTSD and experience its associated emotional, behavioral, cognitive, social, and physical symptoms (Perry, 1999). Another study showed 35% of adolescents with cancer also met criteria for PTSD, with 15% of children surviving cancer still having moderate to severe PTSD (Pelcovitz et al., 1998). Overall, it is clear that traumatic injury or illness may be a cause of comorbid pain and PTSD in children.

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Very few studies have examined the relationship between pain and the course of PTSD. However, Saxe et al. (2001) examined the relationship between pain and the course of PTSD in 24 children who experienced burns, with the aim of investigating the efficacy of using opiate medications (morphine) as a possible preventive agent in children with burn-related PTSD. Results indicated that the dose of morphine administered to these children was associated with a significant reduction of PTSD symptoms over a 6-month period, after controlling for other factors. The authors suggest that acute trauma leads to enhanced fear conditioning and memory consolidation of the trauma and that morphine may have diminished the hyperadrenergic state in these children by inhibiting the fear conditioning and memory consolidation. However, because of the small sample size of children assessed and lack of an experimental design, the authors suggested that future studies should examine more specifically the relationship among opiate dose, noradrenergic function, and PTSD symptoms using a randomized treatment design.

An emerging research question concerns the degree to which comorbid pain and trauma in children interrelate to produce risk for psychopathology. Pine and Cohen (2002) reviewed evidence that suggested that the degree of psychopathology resulting from pain and PTSD in children may depend on the specific form of trauma. For example, compared with children exposed to accidents or natural disasters, children who experience pain as a result of physical or sexual abuse exhibit higher rates of psychopathology. Other factors that may influence differential symptom trajectories in children exposed to traumatic injury include level of trauma exposure, proximity to the trauma, prior trauma exposure, extent of disruption in social support systems, pretrauma levels of psychopathology, child age, gender, and developmental level, and child/parent coping styles (Caffo & Belaise, 2003). Although there are many psychiatric implications after painful injury due to trauma, a review of the literature indicates that PTSD is the most common psychiatric disorder found in children and adolescents. Other comorbid diagnoses, such as mood disorders, anxiety disorders, and conduct disorders, may also occur.

Although no specific developmental models have been proposed to help explain the possible relationship between childhood chronic pain and PTSD, several developmental factors could be important to consider when developing a model that might explain the mutual maintenance of the two disorders. Similar to an adult model described in a previous section of this chapter (i.e., Mutual Maintenance Model; Sharp & Harvey, 2001), pain may serve as a reminder of the trauma in children, triggering an arousal response, leading to avoidance of the cause of pain and any memories of the trauma. Avoidance may be adapted as a way of minimizing pain and any disturbing thoughts of the trauma. Such avoidance can be particularly disruptive to a child, who may be avoiding daily activities such as school or social activities that are

crucial for continuing to meet developmental milestones. Further, a child's avoidance may be reinforced by caregivers, who may have low tolerance for their child's distress or who may have also experienced the trauma themselves. Avoidance can contribute to a child's decreased feelings of self-efficacy related to pain, negative expectations about the ability to cope with pain, increased feelings of distress, and an amplified focus of attention on painful sensations. Cognitive demands caused by symptoms of pain and PTSD could limit the child's use of adaptive coping strategies such as exercise, ignoring pain, and use of positive self-coping statements.

Borrowing from Asmundson et al.'s (2002) proposed shared vulnerability model of chronic pain and PTSD, anxiety sensitivity may also be a predisposing factor contributing to the maintenance of chronic pain and PTSD in children. Children with high levels of anxiety sensitivity may become more fearful in response to physical sensations such as breathlessness or dizziness, thinking that these symptoms could signal doom; thus, these children may avoid activities that trigger physical sensations because of fear of the sensations and of the traumatic cues that they may trigger. These physiological sensations of arousal because of anxiety or PTSD may even be mistaken for being pain related. In fact, in a study of the relationship between anxiety sensitivity and fear of pain in healthy adolescents, anxiety sensitivity was found to account for a unique proportion of the variance in pain anxiety symptoms, even after controlling for other potential predictors of fear of pain (Muris et al., 2001). Although there are studies that have indicated that anxiety sensitivity may play a role in anxiety disorders such as panic in children (Mattis & Pincus, 2003), there are no known studies specifically examining anxiety sensitivity as a potential vulnerability factor for children contributing to the maintenance of chronic pain and PTSD. Thus, the construct of anxiety sensitivity should be explored in future research with this population, and treatments might then be tailored to help children become less fearful of these symptoms through symptom induction techniques such as interoceptive exposure.

The Triple Vulnerability Model, developed by Barlow (2000, 2002) to explain the development of anxiety and panic, may also be relevant to help explain the maintenance and development of psychopathology in children experiencing comorbid chronic pain and PTSD. For example, according to the Triple Vulnerability Model (reviewed earlier), children may have inherited a biological vulnerability to experience psychopathology. Early experiences of lack of control over salient events may be especially relevant for children experiencing trauma and pain (two salient experiences for which the child has little control), contributing to the child's generalized psychological vulnerability. As a result of these early experiences, children may learn to focus their anxiety on specific situations (presumably those that trigger reminders of the pain or trauma). When negative affect and a sense of uncontrollability develop, PTSD and chronic pain conditions may emerge. Development of psychopathology, however, should be moderated to some extent by variables such as the presence of

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"protective factors," including adequate coping skills, social support, and parenting styles that support the child's continued positive growth and development. Both risk and protective factors should be considered in developing a model of the maintenance of pain and PTSD in children.

Developmental psychopathology literature has examined the concept of child resilience and has been concerned with identifying factors associated with increased risk (vulnerability) or decreased risk (resilience) for developing psychopathology after exposure to extreme stress (Masten & Reed, 2002). Such factors should be considered in the development of a model of chronic pain and PTSD in children, rather than simply applying adult models indiscriminately to children. For example, protective factors, or measurable characteristics of a child or situation that predict positive outcome in the context of high risk or adversity, should be incorporated in a theoretical model attempting to explain why some children do not experience high distress and disability from comorbid chronic pain and PTSD, whereas others do. Some common "protective factors" for children that have been identified from a host of studies include good cognitive and coping abilities, positive self-perceptions, faith and meaning in life, close relationships with care-giving adults, an organized home environment, socioeconomic advantages, and close relationships with prosocial and rule-abiding peers (Masten & Reed, 2002). Thus, treatment might focus on increasing the presence of such protective factors in a child's life to maximize chances that children can "bounce back" from the distress caused by chronic pain and PTSD.

9. Implications for Assessment and Treatment of Comorbid Pain and PTSD

Given the high rates of comorbidity of chronic pain and PTSD, clinicians conducting diagnostic assessments of patients with either condition should assess for both disorders, regardless of the age of the patient. There are several well-validated self-report questionnaires that can be used to assist in determining a diagnosis and the severity of symptoms. Self-report measures of pain for adults include the 0 to 10 numerical pain rating scale, the McGill Pain Questionnaire (Melzack, 1975), or the West Haven-Yale Multidimensional Pain Inventory (Kerns et al., 1985). Measures of PTSD for adults include the Posttraumatic Stress Disorder Checklist (Weathers et al., 1993) or the Clinician-Administered PTSD Scale (used to establish a diagnosis of PTSD) (Blake et al., 1990). For a comprehensive review of PTSD assessment measures, see Wilson and Keane (2004). Measures of anxiety sensitivity (Reiss et al., 1986), pain-coping style (Riley et al., 1999), beliefs and expectations related to pain (Jensen et al., 1994), cognitive and behavioral avoidance, and self-efficacy could also be included in the assessment to gain a comprehensive understanding of the factors contributing to and maintaining these conditions.

For children, assessing pain and PTSD can be even more challenging, as age can affect the expression of pain and their ability to report pain or trauma. Typically, below the age of 3 years, children express pain through behavioral manifestation, whereas by the age of 4 years, most children can express pain verbally and indicate the severity of pain using pain-rating scales. The assessment of pain should be sensitive to the child's age, the type of pain, the situation in which pain occurs, the child's prior pain experience, behavioral and emotional factors, and the caregiver's responses and attitudes. There are several child self-report measures of pain, including the Faces Scale (Hicks et al., 2001), the Varni-Thompson Pediatric Pain Questionnaire (Frank et al., 2000), and the Children's Comprehensive Pain Questionnaire (McGrath, 1989). In addition, children may be asked to keep a pain diary. For PTSD, in addition to structured clinical interviews, self-report questionnaires such as the PTSD Reaction Index (Belter et al., 1991), the Children's Posttraumatic Stress Disorder Inventory (Saigh, 1989), or the Impact of Events Scale (Horowitz et al., 1979) are also used. Although it is beyond the scope of this chapter to describe each assessment tool in detail, treatment of children with chronic pain and PTSD should begin with a careful assessment, which should continue throughout treatment.

Only a few studies have reported results of treatments designed to address co-occurring chronic pain and PTSD. Preliminary research suggests that treatment of adults with propranolol following an acute psychologically traumatic event may have a preventative effect on subsequent PTSD; however, further research is needed in this area (Pitman et al., 2002). Muse (1986) described the sequential treatment of three individuals with co-occurring chronic pain and PTSD. Each patient was treated for pain using a behavioral approach that included techniques such as relaxation training, exercise, and biofeedback training. Following treatment for pain, patients were enrolled in a program of systematic desensitization for PTSD. Although the author's discussion of the techniques employed when providing pain treatment and of pain treatment outcomes were vague, the results indicated that subsequent behavioral treatment for PTSD was effective for this population. Of note, treatment in these case studies was sequential; patients were treated first for chronic pain and then for PTSD. In a related study, Shipherd et al. (2003) described a series of case studies that utilized a 12-week manualized PTSD treatment for six women diagnosed with chronic pain and PTSD secondary to MVAs. The treatment included techniques such as imaginal exposure, cognitive restructuring, relaxation techniques, and pleasant activity scheduling. At the 1-month follow-up, patients showed improvements of some PTSD symptoms, but no decrease in pain was noted. Hickling et al. (1992) described 12 patients with posttraumatic headache secondary to a MVA who were effectively treated with CBT. It was reported that the eight participants diagnosed with PTSD failed to show positive results with the headache until the PTSD symptoms were addressed, and they required significantly longer treatment than those who did not meet diagnostic criteria for PTSD. As there

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are no studies that have examined the efficacy of integrated treatments for comorbid pain and PTSD, further investigation of the potential efficacy of an integrated treatment is warranted. As both chronic pain and PTSD respond well to CBT approaches, an integrated CBT protocol may be the most efficient form of treatment.

The theoretical models presented have yet to be fully tested, and further research is needed to determine how clinical treatment protocols should be modified to integrate treatments for both disorders. However, data from existing case studies point toward several techniques that might be useful to utilize when treating a person with comorbid pain and PTSD. The strategies would likely include standard CBT techniques such as cognitive restructuring or coping skills training, education about the function of avoidance, and ways to conduct situational exposures (e.g., doing activities that were previously avoided), as well as interoceptive exposures (e.g., exercises used to bring on physical sensations). Patients could also be taught to reinforce positive self-efficacy beliefs, correct attentional biases, and reduce catastrophizing. Overall, it is important to help patients with pain and PTSD understand the ways these two disorders may be maintained and the importance of decreasing avoidance. As patients begin to increase their participation in daily activities, they may be better able to obtain a more positive quality of life.

10. Future Directions

The research summarized in this review suggests that chronic pain and PTSD frequently co-occur throughout one's life span, and that similar mechanisms, such as fear and avoidance, anxiety sensitivity, and catastrophizing, as well as general and specific psychological vulnerabilities, may help account for the development and maintenance of both conditions. Although several models have been proposed to explain the relationship between chronic pain and PTSD, continued research in this area is needed to more fully develop and test these models. Several factors have been presented that might be important to include in the conceptualization of a model of pain and PTSD in childhood, so that adult models are not indiscriminately applied to children without considering developmental factors. Regardless of patient age, given the high rate at which chronic pain and PTSD co-occur, it is imperative that researchers develop more integrated assessment and treatment techniques for this population. In pursuit of this goal, a study recently funded by the Department of Veterans Affairs Rehabilitation Research and Development Service is being conducted by Otis and Keane at the VA Boston Healthcare System to examine the efficacy of an integrated cognitive-behavioral treatment program for veterans with comorbid chronic pain and PTSD; the study will also examine potential mechanisms of action that may explain the relationship between chronic pain and PTSD. As such studies are launched and treatment strategies are tested with patients of all ages with comorbid pain

and PTSD, we may then begin to refine our existing treatment protocols. This can only be done through systematic and well-controlled research.

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